JACC Vol. 15, No. 2
February 1990:426–8

Editorial Comment

"Cutters, Scoopers, Shavers and Scrapers": The Importance of Atherectomy Devices and Clinical Relevance of Tissue Removed*

BRUCE F. WALLER, MD, FACC,
CASS A. PINKERTON, MD, FACC

Indianapolis, Indiana

Many interventional devices are currently part of our armamentarium for the treatment of obstructed arteries (1). Some of these devices result in *remodeling* of the obstructing material, whereas others alter luminal obstruction by *removing* the tissue rather than displacing, cracking, crushing or compressing it. One of the exciting new tools in the area of tissue removal is the atherectomy device. The prototype of this device is the Simpson atherotome consisting of a motor-driven cutter rotating at 2,000 rpm that slowly "shaves away" or "scoops out" obstructing material. The excised material is deposited in a distal collection chamber.

The present study. In this issue of the Journal, Johnson et al. (2) report the first histologic observations on excised peripheral arterial tissue from transluminal atherectomy procedures in 100 patients. Three major categories of tissue were found: 1) atherosclerotic plaque (with or without thrombus), 2) intimal fibrous proliferation (fibrous intimal hyperplasia), and 3) thrombus only. Of *native* lesions treated with the atherectomy device, 88% of the tissue samples were typical atherosclerotic plaque, 9% were fibrous intimal thickening with medial calcinosis and 3% consisted of thrombus only. Among *postinterventional lesions* (restenosis after primary balloon angioplasty or primary atherectomy, or both) treated with subsequent atherectomy, 75% of those excised consisted of intimal fibrous proliferation and 25% were classic atherosclerotic plaque identical to samples recovered from native lesions.

Conclusions derived from the study. At least four clinically relevant observations are apparent from the morphologic-histologic data presented by Johnson et al. (2):

1. *Intimal fibrous proliferation ("restenosis") lesions reported in necropsy patients with preceding balloon angioplasty procedures are histologically identical to those in living patients.* In the past, some investigators have suggested that the intimal fibrous hyperplasic lesions of restenosis might be unique to necropsy patients. Johnson et al. (2) are the first to document the identical nature of angioplasty restenosis tissue in living and in necropsy patients.

2. *Clinical and angiographic restenosis occurs after primary atherectomy procedures.* As a potential alternative to balloon angioplasty or as a complementary method of therapy, transluminal atherectomy does not appear to be free of the problem of restenosis. In the study by Johnson et al. (2), 16 (18%) of 89 patients initially treated with atherectomy returned with restenosis, and 3 (27%) of 11 patients treated with atherectomy for restenosis after previous balloon angioplasty returned with a second restenosis.

3. *Restenosis lesions occurring after balloon angioplasty do not consist exclusively of intimal fibrous proliferation tissue.* At least 25% of the restenosis tissue removed by transluminal atherectomy is atherosclerotic plaque indistinguishable from the native lesion (Fig. 1). Atherectomy specimens from restenosis lesions consisting only of atherosclerotic plaque can be explained in several ways: 1) the initial stenotic atherosclerotic lesion was inadequately, insufficiently or superficially dilated; 2) the initial angioplasty mechanism was concentric or eccentric vessel stretching; 3) the restenosis lesion represents accelerated development of atherosclerotic plaque; or 4) various combinations of these conditions exist. Accelerated development of atherosclerotic plaque at the site of restenosis seems an unlikely explanation, because the time interval from the interventional procedure to restenosis is only a few months and the restenosis material consists of densely fibrotic and calcified material (i.e., "old plaque"). Different morphologic substrates for restenosis (atherosclerotic plaque, intimal fibrous proliferation, stretching of eccentric lesions) suggest that *different* treatment strategies may be necessary for each type of restenosis lesion.

4. *Arterial vessel healing after angioplasty or atherectomy, or both, is nonspecific and appears to be independent of the mechanism by which the arterial injury or disruption initially occurred.* Johnson et al. (2) compared the intimal fibrous proliferation material at restenosis sites from previous primary balloon angioplasty and previous primary atherectomy procedures and found them histologically similar in cellularity, vascularity, degree of fibrosis, content of inflammatory cells and presence of thrombus. Furthermore, four restenosis lesions in which both angioplasty and atherectomy were the primary interventional therapy showed no histologic differences from those of either proce-

*Editorials published in *Journal of the American College of Cardiology* reflect the views of the authors and do not necessarily represent the views of JACC or the American College of Cardiology.

From Nasser, Smith, Pinkerton Cardiology, Inc., The Indiana Heart Institute, and the Departments of Medicine and Pathology, St. Vincent Hospital, Indianapolis, Indiana.

Address for reprints: Bruce F. Waller, MD, Suite 400, St. Vincent Professional Building, 8402 Harcourt Road, Indianapolis, Indiana 46260.

0735-1097/90/$3.50

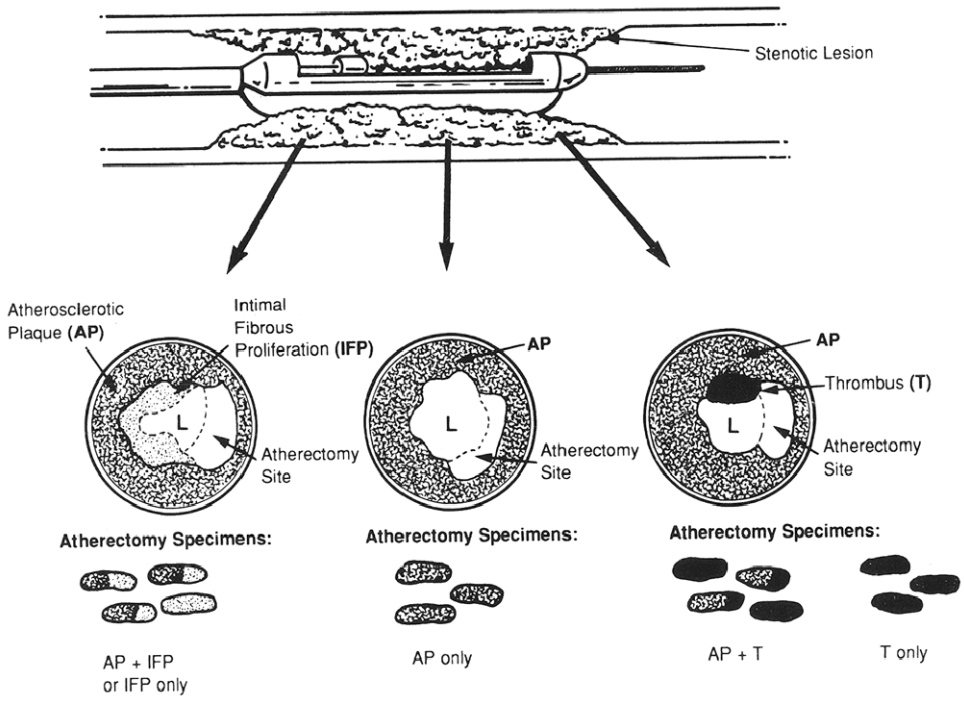

Figure 1. Possible morphologic explanations for "restenosis" material removed by transluminal atherectomy devices. Atherectomy specimens consist of combined atherosclerotic plaque with intimal fibrous proliferation, intimal fibrous proliferation only, atherosclerotic plaque only, atherosclerotic plaque with thrombus or thrombus only. The **dashed line** indicates the original luminal surface before atherectomy. L = lumen.

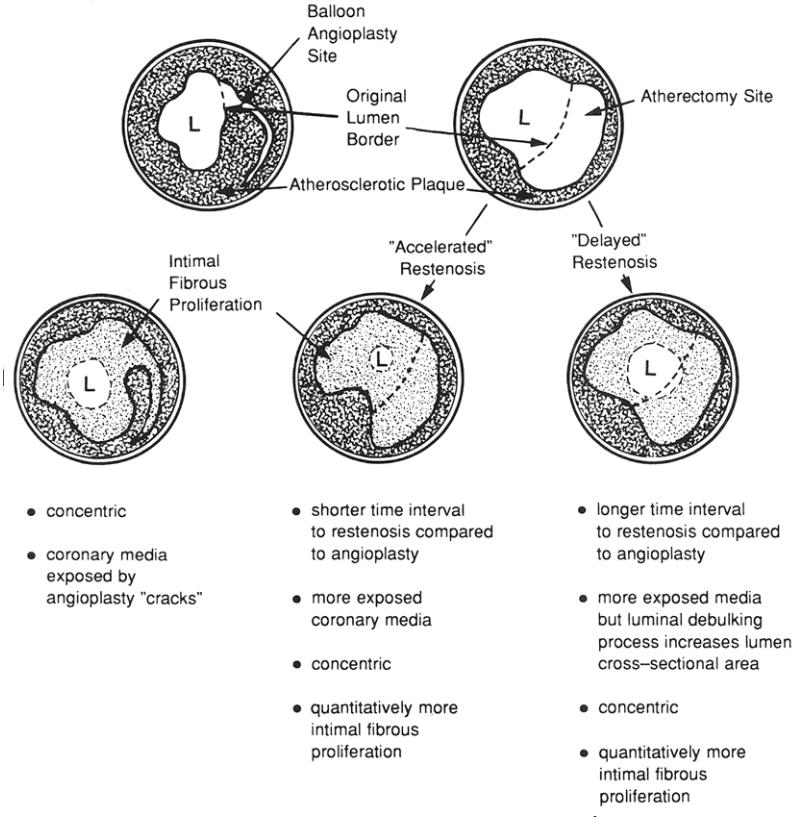

Figure 2. Morphologic comparison of restenosis lesions following primary transluminal balloon angioplasty versus primary transluminal atherectomy. Restenosis following atherectomy may have a similar, shorter ("accelerated") or longer ("delayed," "reset") interval from intervention to clinical restenosis. The restenosis tissue in either intervention is intimal fibrous proliferation. The **dashed line** indicates the original luminal surface before atherectomy. L = lumen.

JACC Vol. 15, No. 2
February 1990:426–8

dure alone. Thus, it appears that angioplasty, atherectomy and perhaps other interventional procedures associated with intimal fibrous proliferation restenosis could have a *common* method of treatment or prevention because the morphologic response to vessel injury is the same.

Unresolved issues. Although the report by Johnson et al. (2) addresses several topics of restenosis and atherectomy procedures, many issues concerning restenosis following atherectomy procedures remain unresolved: Is the interval of time from the atherectomy procedure to clinical restenosis, similar to or shorter or longer than that of primary balloon angioplasty? How much arterial media is removed at atherectomy? Does the exposure of more arterial media during atherectomy procedures than during angioplasty procedures result in "accelerated" restenosis (Fig. 2)? Does increased luminal cross-sectional area created by the actual removal of obstructing native lesions override the potentially adverse effect of greater medial layer exposure and, thus, "delay" or "reset" the timing of clinical restenosis (Fig. 2)? These and other questions will be the subject of future studies.

Conclusions. The clinical usefulness of the atherectomy procedure is not limited to its role in removal of obstructing material in symptomatic patients. Atherectomy can be a tool in obtaining *human* restenosis tissue for tissue culture or for the preparation of monoclonal blocking agents to smooth muscle cells or other "specific restenosis cells." It can aid in evaluating differences in restenosis tissue after various pharmacologic therapies for preventing restenosis. It can also be a tool for the removal of large intimal flaps or occluding thrombus in acute or abrupt occlusions after balloon angioplasty.

Johnson et al. (2) are to be congratulated on their report and for providing new insights into the processes of restenosis after angioplasty and raising new and challenging questions for future research with transluminal atherectomy.

References

1. Waller BF. "Crackers, breakers, stretchers, drillers, scrapers, shavers, burners, welders and melters"—the future treatment of atherosclerotic coronary artery disease? A clinical-morphologic assessment. J Am Coll Cardiol 1989;13:969–87.

2. Johnson DE, Hinohara T, Selmon MR, Braden LJ, Simpson JB. Primary peripheral arterial stenoses and restenoses excised by transluminal atherectomy: a histopathologic study. J Am Coll Cardiol 1990;15:419–25.